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L2   ANSWER 1 OF 2     MEDLINE on STN                    DUPLICATE 1
AN   2001664325      MEDLINE
DN   PubMed ID: 11518704
TI   IRAK-mediated translocation of TRAF6 and TAB2 in the interleukin-1-induced
     activation of NFkappa B.
AU   Qian Y; Commane M; Ninomiya-Tsuji J; Matsumoto K; Li X
CS   Department of Immunology, Lerner Research Institute, the Cleveland Clinic
     Foundation, Cleveland, Ohio 44195, USA.
NC   GM 600020 (NIGMS)
SO   Journal of biological chemistry, (2001 Nov 9) 276 (45) 41661-7.
     Journal code: 2985121R. ISSN: 0021-9258.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200112
ED   Entered STN: 20011119
     Last Updated on STN: 20030105
     Entered Medline: 20011205
AB   The interleukin-1 (IL-1) receptor-associated kinase (IRAK) is required for
     the IL-1-induced activation of nuclear factor kappaB and c-Jun N-terminal
     kinase.  The goal of this study was to understand how IRAK activates the
     intermediate proteins TRAF6, TAK1, TAB1, and TAB2.  When IRAK is
     phosphorylated in response to IL-1, it binds to the membrane where it
     forms a complex with TRAF6; TRAF6 then dissociates and translocates to the
     cytosol.  The membrane-bound IRAK similarly mediates the IL-1-induced
     translocation of TAB2 from the membrane to the cytosol.  Different regions
     of IRAK are required for the translocation of TAB2 and TRAF6, suggesting
     that IRAK mediates the translocation of each protein separately.  The
     translocation of TAB2 and TRAF6 is needed to form a TRAF6-TAK1-TAB1-TAB2
     complex in the cytosol and thus activate TAK1.  Our results show that IRAK
     is required for the IL-1-induced phosphorylation of
     TAK1, TAB1, and TAB2.  The phosphorylation of these
     three proteins correlates strongly with the activation of nuclear factor
     kappaB but is not necessary to activate c-Jun N-terminal kinase.

L2   ANSWER 2 OF 2     MEDLINE on STN                    DUPLICATE 2
AN   2000167218      MEDLINE
DN   PubMed ID: 10702308
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TI TAK1 mitogen-activated protein kinase kinase kinase is activated by autophosphorylation within its activation loop.

AU Kishimoto K; Matsumoto K; Ninomiya-Tsuji J

CS Department of Molecular Biology, Graduate School of Science, Nagoya University and CREST, Japan Science and Technology Corporation, Chikusa-ku, Nagoya 464-8602, Japan.

SO Journal of biological chemistry, (2000 Mar 10) 275 (10) 7359-64. Journal code: 2985121R. ISSN: 0021-9258.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200004

ED Entered STN: 20000413
Last Updated on STN: 20000413
Entered Medline: 20000403

AB TAK1, a member of the mitogen-activated kinase kinase kinase family, is activated in vivo by various cytokines, including interleukin-1 (**IL-1**), or when ectopically expressed together with the **TAK1**-binding protein **TAB1**. However, this molecular mechanism of activation is not yet understood. We show here that endogenous TAK1 is constitutively associated with TAB1 and phosphorylated following IL-1 stimulation. Furthermore, TAK1 is constitutively phosphorylated when ectopically overexpressed with TAB1. In both cases, dephosphorylation of TAK1 renders it inactive, but it can be reactivated by preincubation with ATP. A mutant of TAK1 that lacks kinase activity is not phosphorylated either following **IL-1** treatment or when coexpressed with **TAB1**, indicating that **TAK1** phosphorylation is due to autophosphorylation. Furthermore, mutation to alanine of a conserved serine residue (Ser-192) in the activation loop between kinase domains VII and VIII abolishes both phosphorylation and activation of TAK1. These results suggest that **IL-1** and ectopic expression of **TAB1** both activate **TAK1** via autophosphorylation of Ser-192.

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1: Cheung PC, Campbell DG, Nebreda AR, Cohen P. Related Articles,

Feedback control of the protein kinase TAK1 by SAPK2a/p38alpha.
EMBO J. 2003 Nov 3;22(21):5793-805.
PMID: 14592977 [PubMed - indexed for MEDLINE]

2: Jang SB, Won J, Kim H, Kim J, Lee KH, Han H, Rha HK, Choi CR. Related Articles,

TAK1 mediates lipopolysaccharide-induced RANTES promoter activation i BV-2 microglial cells.
Mol Cells. 2002 Aug 31;14(1):35-42.
PMID: 12243350 [PubMed - indexed for MEDLINE]

3: Kawahara T, Kuwano Y, Teshima-Kondo S, Sugiyama T, Kawai T, Nikawa T, Kishi K, Rokutan K. Related Articles,

Helicobacter pylori lipopolysaccharide from type I, but not type II strains, stimulates apoptosis of cultured gastric mucosal cells.
J Med Invest. 2001 Aug;48(3-4):167-74.
PMID: 11694956 [PubMed - indexed for MEDLINE]

4: Kawahara T, Teshima S, Kuwano Y, Oka A, Kishi K, Rokutan K. Related Articles,

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Am J Physiol Gastrointest Liver Physiol. 2001 Sep;281(3):G726-34.
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5: Wang C, Deng L, Hong M, Akkaraju GR, Inoue J, Chen ZJ. Related Articles,

TAK1 is a ubiquitin-dependent kinase of MKK and IKK.
Nature. 2001 Jul 19;412(6844):346-51.
PMID: 11460167 [PubMed - indexed for MEDLINE]

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Items 1 - 11 of 11 One

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TAB2 and TAB3 activate the NF-kappaB pathway through binding to polyubiquitin chains.
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PMID: 15327770 [PubMed - indexed for MEDLINE]

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Feedback control of the protein kinase TAK1 by SAPK2a/p38alpha.
EMBO J. 2003 Nov 3;22(21):5793-805.
PMID: 14592977 [PubMed - indexed for MEDLINE]

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Vet Immunol Immunopathol. 2003 Jun 20;93(3-4):177-84.
PMID: 12814703 [PubMed - indexed for MEDLINE]

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Cytokines suppress adipogenesis and PPAR-gamma function through the TAK1/TAB1/NIK cascade.
Nat Cell Biol. 2003 Mar;5(3):224-30.
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TAB2 is essential for prevention of apoptosis in fetal liver but not for interleukin-1 signaling.
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IRAK and TAK1 are required for IL-18-mediated signaling.
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IRAK-mediated translocation of TRAF6 and TAB2 in the interleukin-1-indu activation of NFkappa B.
J Biol Chem. 2001 Nov 9;276(45):41661-7. Epub 2001 Aug 22.
PMID: 11518704 [PubMed - indexed for MEDLINE]

9: Wang C, Deng L, Hong M, Akkaraju GR, Inoue J, Chen ZJ. Related Articles,

TAK1 is a ubiquitin-dependent kinase of MKK and IKK.
Nature. 2001 Jul 19;412(6844):346-51.
PMID: 11460167 [PubMed - indexed for MEDLINE]

10: Holtmann H, Enninga J, Kalble S, Thiefes A, Dorrie A, Broemer M, Winzen R, Wilhelm A, Ninomiya-Tsuji J, Matsumoto K, Resch K, Kracht M. Related Articles,

The MAPK kinase kinase TAK1 plays a central role in coupling the interle 1 receptor to both transcriptional and RNA-targeted mechanisms of gene regulation.
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PMID: 10702308 [PubMed - indexed for MEDLINE]

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L2	0	(tak1 near2 tab1) near10 LPS	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 17:13